11:15

782-4 Analysis of Heat Generation During High-Speed Rotational Ablation: Technical Implications

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To date no data has correlated aggressive advancement of the Rotablator® measured by significant drops in speed with heat generation. We analyzed the impact of Rotablator technique on temperature change (ΔT) in two experimental models. Burrs of 1.75 mm advanced through 0.35 mm lumens generated in 12 mm cylinders of bovine bone that were continuously flushed with saline. Measurements were performed with 4 implanted thermal probes separated by 3 mm intervals. The following techniques were compared: continuous ablation (CA) vs. intermittent ablation (IA) with excessive deceleration of 10–20 K (ED) or minimal deceleration –5–10 K (MD). The following results were obtained.

°C	CA-ED	IA-ED	CA-MD	IA-MD*
ΔT ± sd (n = 4)	4.8 ± 1.7	2.2 ± 2.5	5.7 ± 1.4	0.6 ± 0.9

*significantly less p < 0.01 when compared to CA-ED and CA-MD

Second, multiple polytetrafloroethylene grafts were used to segmentally constrict porcine femoral arteries. An oversized burr (2.25 mm) was advanced through the segment, and Δ T was recorded with thermal probes. With RPM decreases of 2–9 K the temperature rose 3°C ± 1.4 while RPM drops of 10–15 K resulted in a 10°C ± 6.8 temperature increase. *Conclusion:* Excessive drops in speed and aggressive advancement of the burr are related to substantial increases in temperature and potential thermal injury. The acute and long term effects will be the subject of further investigation.

11:30

782-5 Is There a Role for Strip Chart Recording to Guide Rotational Atherectomy? Initial Findings From Stratas

David S. Eccleston, Mark C. Horrigan, Michael J. Cowley, Richard E. Kuntz, David O. Williams, Patrick Whitlow, for the STRATAS Investigators. *Cleveland Clinic Foundation, Cleveland, Ohio*

Study To Determine Rotablator and Transluminal Angioplasty Strategy (STRATAS) is a multicenter randomized trial of routine (burr/artery 0.5–0.7 with adjunctive PTCA) and aggressive debulking strategies (burr/artery 0.7–0.9, alone or with PTCA ≤ 1 atm) to determine which approach optimizes clinical outcomes and minimal luminal diameter at 6 months. During rotational atherectomy, strip-chart recordings are made plotting burr speed against time to allow investigation of potential relationships between decelerations of 5–10,000 r.p.m. and procedural complications or restenosis. To date, 78 patients have been enrolled; demographic and strip chart data are presented for the first fifty. Mean age was 64 ± 12 years, 68% of patients were male, 14% current smokers and 23% diabetic, and mean left ventricular ejection fraction 62 ± 10%. Strip chart data are tabulated below.

Variable	Mean ± S.D. (n = 50)
No. of decelerations > 5000 rpm	8.3 ± 8.3
Rotablator time (sec)	381 ± 212
Total deceleration time (sec.)	11.3 ± 11.5
No. of decelerations/minute	1.5 ± 1.7
% Time decelerated > 5000 rpm	3.6 ± 4.5

The procedural success rate (residual stenosis < 50% without death, Q-wave infarction or emergency bypass surgery) was 95.8%, with no complications in 87.2%. All 3 patients with Q-wave infarction or CK > 8× normal had decelerations > 7000 rpm for ≥ 5 seconds, and 2/4 with CK 3–8× normal had decelerations of this magnitude.

Conclusion: Strip chart recordings are useful in guiding rotational atherectomy technique and may have utility in predicting adverse 1 outcomes.

11:45

782-6 Different Propensity of Coronary Restenosis: Comparison Between Cutting Balloon, Conventional Balloon and Atherectomy

Raoul Bonan, Olivier F. Bertrand, Alan Adelman, Michel Joyal, Christina Utenberg, Andreas Zelher, Jean-Marc Lablanche, Michel Bertrand, Peter de Jaegere, Peter Ruygrok. *Montreal Heart Institute Quebec, Canada*

Restenosis after coronary dilatation still remains an unresolved question and involves several biological processes as myointimal proliferation and vessel remodeling. Absolute diameters measured by quantitative angiography (QCA) have been proposed to characterize more adequately this continuum process. To analyse long term angiographic outcome after Cutting Balloon (CB) dilatation (3–4 blades scoring the plaque, low-pressure dilatation ≤ 8 atm, short inflation time ≤ 90 s), we compared absolute and relative angiographic parameters with those obtained in the Canadian Coronary Atherectomy Trial (CCAT).

	CB	CB + PTCA	PTCA	DCA
Pts	78	42	123	138
LAD (%)	42	43	100	100
Ref (mm)	2.80 ± 0.44	2.85 ± 0.41	3.21 ± 0.48*	3.14 ± 0.47*
A.gain (mm)	0.70 ± 0.33	0.90 ± 0.33°	1.15 ± 0.44°°	1.43 ± 0.46°°°
L.Loss(mm)	0.23 ± 0.39	0.39 ± 0.47	0.47 ± 0.6′	0.78 ± 0.6″
Loss index	0.32 ± 1.06	0.61 ± 1.3	0.43 ± 0.8	0.57 ± 0.47#

*p < 0.001 vs CB, CB + PTCA °p < 0.01 vs CB, °°p < 0.001 vs CB, CB + PTCA, °°°p < 0.001 vs CB, CB + PTCA, ′p < 0.005 vs CB, ″p < 0.001 vs CB, CB + PTCA, #p < 0.05 vs CB

The reduction of the traumatism to the artery wall by scoring and dilating at low pressure with the CB, in accordance with animal experimental studies does produce less luminal loss at 6 month follow-up and seems to give a better loss index. Ongoing randomized trial with conventional angioplasty will evaluate the direct impact of the CB on clinical and angiographic restenosis.

783 Myocardial Infarction – Basic Mechanisms – Ischemic Preconditioning and Remodeling

Wednesday, March 27, 1996, 10:30 a.m.–Noon
Orange County Convention Center, Room 315

10:30

783-1 Two Distinct Mechanisms Induce a Subtype-Selective Activation of Protein Kinase C in Acute Myocardial Ischemia

Ruth H. Strasser, Martin Braun, Steffen H. Schön, Annette Kempkes, Renate Ihl-Vahl, Rainer Marquetant. *University of Heidelberg, Germany*

Protein kinase C (PKC) plays an important role in acute myocardial ischemia and ischemic remodeling and it may be crucially involved in ischemic preconditioning. Acute myocardial ischemia (≤ 15 min) has been shown to promote a rapid translocation to the plasma membranes and activation of all predominant cardiac isoforms of PKC. In contrast, prolonged ischemia (≥ 30 min) induces an increased enzyme activity in the cytosol, but not in the plasma membranes. This late regulation promotes increased levels of the isoform PKC-ε and -δ, but not of PKC-α and -ζ as demonstrated by quantitative Western blot analysis. To investigate, if this regulation of PKC in prolonged ischemia may be mediated by an expressional control we determined the steady state mRNA-levels specific for all cardiac isoforms using quantitative RT-PCR. For absolute quantification isoform-specific cRNA standards were used. Specific PCR-products were quantified using internal oligonucleotides in Southern blot hybridizations.

In isolated perfused rat hearts prolonged ischemia (≥ 30 min) induces a subtype-selective increase of the mRNA-levels for PKC-ε by 69% (69 ± 7.1 vs. 118 ± 9.7 ag/ng total RNA) and for PKC-δ by 76% (35 ± 3.9 vs. 62 ± 3 ag/ng total RNA). In contrast, the mRNA-levels for PKC-α and -ζ ranging at about 20 ag/ng total RNA remained unchanged.

These data characterize for the first time a new mechanism for the subtype-selective regulation of PKC isoforms in prolonged ischemia. This expressional regulation results in increased mRNA-levels selectively for PKC-ε and PKC-δ leading to augmented enzyme levels. This regulation contrasts the rapid translocation and activation of the constutively expressed PKC isoforms in the early phase of ischemia. Further studies have to evaluate which role this newly characterized, subtype-selective expressional regulation of PKC-isoforms may play in the remodeling process of the infarcted heart.

10:45

783-2 Brief Intracoronary Calcium Infusion "Preconditions" Canine Myocardium

Karin Przyklenk, Katsuya Hata, Robert A. Kloner. *Heart Institute, Good Samaritan Hospital and USC, Los Angeles CA*

The cellular mechanism by which brief ischemia "preconditions" the heart and renders the myocardium resistant to subsequent sustained coronary occlusion (CO) remains unresolved. We propose that a transient increase in intracellular calcium (Ca) concentration — a well-documented consequence of brief nonlethal ischemia — may be an important component of the second messenger pathway responsible for the reduction of infarct size seen with preconditioning. If so, then brief intracoronary (i.c.) infusion of Ca should,